already reduced cardiac output from cardiac dysrhythmias.

Acute increases in serum potassium can lead to cardiac conduction block and arrest. Acute hyperkalemia may be caused by inappropriate exogenous administration of potassium chloride or the use of succinylcholine for muscle relaxation in patients who are severely burned, traumatized or have spinal cord injuries. When the serum potassium is allowed to accumulate in the body from lack of elimination (that is, renal failure), the potassium ions tend to distribute in body compartments maintaining the normal transmembrane potassium concentration gradient. Therefore, in chronically hyperkalemic patients elevated serum potassium levels tend to be tolerated far better than in normal patients whose serum potassium concentration is subjected to an acute rise. Nevertheless, a serum potassium value greater than 10 mEq per liter usually leads to cardiac arrest regardless of whether the hyperkalemia is acute or chronic.

In summary, both hypokalemia and hyperkalemia can disturb electrophysiology and induce cardiac failure. It is important to accurately determine the cause of the serum potassium abnormality in order to institute appropriate management.

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Local Anesthetic Agents in Obstetrics—An Update

THE USE OF local anesthetic agents to relieve the discomfort of the parturient during labor and delivery is common practice. We have in our armamentarium a choice of paracervical, paravertebral, epidural or caudal block to provide analgesia during labor, and we may utilize pudendal, subarachnoid (saddle), epidural, caudal or field block to provide perineal anesthesia for delivery. In the last decade there have been major advances in our understanding of the effects of local anesthetics on both fetuses and neonates. For example, we now realize that local anesthetics may readily cross the placenta and produce undesirable responses in a fetus. Second, more sophisticated methods are available to study the subacute effects of local anesthetics on neonatal neurobehavioral function.

The initial interest in fetal and neonatal blood levels of local anesthetics was generated by studies documenting the occurrence of fetal bradycardia following paracervical block and a possible correlation of this event with blood levels of local anesthetics. Subsequent evidence suggests that the mechanism of fetal bradycardia associated with this procedure may be hypoxia caused by local anesthetic-induced uterine artery vasoconstriction. Consequently, paracervical block is losing popularity, and most recent investigations are concerned with epidural and caudal anesthesia. With these latter blocks, significant amounts of local anesthetic can be absorbed by the engorged epidural venous plexus of pregnancy. Similarly, pudendal block and local infiltration of the perineum may also result in significant uptake of local anesthetic into the maternal bloodstream due to the vascularity of the area. Moderate caution should be applied to these techniques.

When selecting an amide-type of local anesthetic agent requiring slow hepatic degradation, consideration should be given to those properties of the drug that allow for transfer from the maternal circulation across the placental membrane to the fetus. In addition, attention should be paid to the disposition and metabolism by the fetus and neonate because this determines ultimate toxicity. Substances that are highly bound to plasma proteins have less free drug available for transfer. Highly ionized or charged compounds do not cross membranes easily. Drugs which are highly lipid-soluble do cross membranes easily, but this same quality may allow for deposition in epidural adipose tissue, thereby decreasing the maternal level. In addition, because of rapid penetration into fetal fat compartments, the fetal blood level is also decreased.

The duration of action is important because the longer-acting agents tend to decrease the total quantity of drug required. Lidocaine and mepivacaine are considered intermediate in action with a duration of 45 to 75 minutes, while the long-acting bupivacaine (Marcaine) has a usual duration of 1½ to 2 hours. The short-acting ester 2-chloroprocaine (Nesacaine) has an average duration of 45 minutes. An additional advantage is its rapid rate of destruction by plasma cholinesterase and a half-life of 21 seconds. Little if any will be available for placental transfer. The

fetus also has plasma cholinesterase activity present as early as 28 weeks of gestation.

The neurobehavioral assessment scale, for use in evaluating subtle neonatal behavioral alterations induced by anesthetic drugs and the events of labor, was introduced in 1973 by Brazelton and recently modified by Scanlon. This complex system allows far more accurate assessment than by Apgar scoring alone. Neurobehavioral assessment of infants of mothers who received local anesthetic drugs in epidural anesthetics for labor and delivery shows that when lidocaine and mepivacaine were used the infants had poorer muscle tone scores and habituation to pinprick at 2 and 4 hours of age. With the use of bupivacaine or 2-chloroprocaine, scores were equal to or better than those in control infants. It is important to note, however, that all infants were the same by the second day of life. No long-term inferences can be made at this time.

The current recommendations for use of local anesthetic drugs in regional anesthesia for obstetrics are to utilize the minimum amount of drug needed to produce the desired effect and to avoid those agents and techniques that cause measurable changes in the fetus or the neonate. Based on the foregoing considerations, epidural or caudal anesthesia with 2-chloroprocaine or bupivacaine (or both) fulfills these requirements.

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Management of Patients With Increased Intracranial Volume or Pressure

THE CRANIAL VAULT is a semi-closed cavity containing brain, blood and cerebrospinal fluid. These three components do not under normal circumstances completely fill the cavity. Rather, there is room available for expansion. Intracranial pressure (ICP) is normally less than 10 torr. ICP does not rise until increases in cranial contents have completely filled the cranial vault. Then, even slight increases in intracranial volume cause steep increases in ICP. Common causes of an increase in volume are brain tumors, posttraumatic swelling, vasodilatation with increased cerebral blood flow or blockage of cerebrospinal fluid drainage. As ICP rises, a critical lowering of the cerebral perfusion pressure can occur and produce cerebral hypoxia. The mean arterial pressure minus the ICP is the cerebral perfusion pressure. There are compensatory mechanisms that can blunt or reverse elevations in ICP. Compensation can occur through cerebral vasoconstriction, brain shrinkage, reduction of cerebrospinal fluid production and relocation of cerebrospinal fluid in the spinal canal.

The treatment of patients with increased intracranial volume makes use of these compensatory mechanisms and must prevent further increases in volume. Respiratory obstruction or depression must be immediately corrected. Carbon dioxide retention produces cerebral vasodilatation as does hypoxemia if arterial oxygen tension falls below 50 torr. Dilatation from hypoxemia is more serious and troublesome than carbon dioxide retention due to its persistence after the hypoxemia has been corrected. Vasodilatation from carbon dioxide retention increases cerebral blood flow 1 ml per 100 grams of brain tissue per minute for each 1 torr rise in arterial carbon dioxide pressure. However, hyperventilation to produce hypocapnia is immediately beneficial as a reduction of cerebral blood flow of 1 ml per minute is achieved with each 1 torr decrease in carbon dioxide pressure. Additional increases in brain volume can be prevented by fluid restriction and the avoidance of 5 percent dextrose in water solutions. Dextrose in water is rapidly absorbed into brain cells carrying water with it. Stabilization of the blood-brain barrier with large doses of steroids, usually dexamethasone, also inhibits swelling. Brain bulk can be reduced through cerebral dehydration with osmotic and renal diuretics such as mannitol and furosemide. Methods of decreasing cerebrospinal fluid production are less well defined, although steroids may decrease production.

Reduction of cerebrospinal fluid volume should only be done at surgical operation. Spinal taps are to be condemned because danger of herniation of the brain is real and little helpful information is obtained. During surgical operation, the head-up position and avoidance of jugular vein compression enhance venous return. Volatile anesthetic agents cause cerebral vasodilatation proportional to their anesthetic depth and, therefore, should be avoided or administered at very light levels only after hypocapnia has been established and verified. Monitoring of arterial blood gases and